

fried in grease and no pies or other pastry. They must eat largely of cereals, milk, fruits, eggs and butter. Of drugs, the fluid extract of horse-nettle berries has given satisfaction, since it does not impair the functions of the digestive tract. Simulo, also, has given excellent results. A combination of bromide, chloral and morphine is often of service in aborting an expected seizure preceded by a long aura. Great stress is to be laid upon the systematic exercise of all the muscles of the patient's body, such work as can best be done being prescribed after a thorough physical examination.

JELLIFFE.

144 THE ETIOLOGY AND PATHOLOGY OF MAJOR EPILEPSY. William House (Phila. Med. Jour., Vol. 5, 1900, p. 691).

In this paper the writer reviews briefly the various theories advanced by different observers as to the causation and pathology of epilepsy, and, believing that "like causes produce like effects," compares the symptoms of epilepsy with those of other diseases which produce or are accompanied by convulsive seizures resembling more or less the seizures of epilepsy. These diseases are hysteria, tetany, infantile, puerperal and uremic eclampsia, alcoholism, cerebral hemorrhage, and the apoplectiform and epileptiform convulsions of general paresis. The pathology of the three latter conditions is well known, and in certain respects the lesions are of a similar nature. The symptoms of an epileptic seizure, of an epileptiform parietic convulsion, of an alcoholic convulsion, and of cerebral hemorrhage present a marked similarity, so much so that sometimes it is difficult to make a differential diagnosis. Symptoms of cerebral pressure are present in these three conditions and also in epilepsy. In the brain of an alcoholic there is an excessive quantity of cerebrospinal fluid, the ventricles are distended, the brain substance drips with fluid, and the membranes are dropsical. This is called the "wet brain." In general paresis the ventricles are distended with fluid, there is an increased quantity of fluid in the subdural space, and the whole brain is surrounded with an excessive quantity of turbid cerebrospinal fluid. In both these conditions the excessive fluid seems to the writer to be the logical cause of the pressure symptoms recognized in convulsions from these diseases.

The writer has witnessed the autopsies of five cases of *status epilepticus*, and in each instance there was found an excessive quantity of cerebrospinal fluid.

Arguing from analogy, House is forced to the conviction that this increase in the quantity of cerebrospinal fluid must bear a causal relation to the convulsions of epilepsy. According to physiologic findings, if the cerebrospinal fluid be suddenly withdrawn convulsions may ensue; if rapidly increased coma may be produced.

From the observation of over 200 epileptics, and from the comparison of their symptoms with those of 60 alcoholics and those of a large number of paretics, from the findings in the above five autopsies, and from an analysis of recent literature, he concludes that:

(1) There is no record of pathologic findings which logically explain the symptoms of epilepsy.

(2) An increase of cerebrospinal fluid would readily account for the seizures. In many instances it is analogous to the marked increase of fluid in the crania of alcoholics and paretics, and is not dissimilar in clinical effects to the more localized lesions of hemorrhage or abscess.

(3) This fluid, physiologically subject to more or less variation in quantity from day to day, is fully capable of pathologic increase, and from analogy must bear exciting relation to the convulsion.

(4) Its increase is probably gradual, and to this we may ascribe the *auræ*.

(5) Its absorption probably begins with the third stage of the convulsion (relaxation and coma), and if this fails, repeated convulsions (*status epilepticus*) ensue.

(6) Its superabundance may be due to lymphatic spasm, or to marked disturbance of equilibrium between lymphatic and general circulatory activity, which may be favored by heredity, toxemia, or any of the recognized predisposing causes.

(7) This creed applies to the so-called idiopathic epilepsy, as distinguished from the convulsion of the Jacksonian epilepsy, although even in such cases this condition will help to explain some otherwise unexplained symptoms. BONAR.

145 ZUR KENNTNISS DER PROGRESSIVEN MUSKELATROPHIE (On Progressive Muscular Atrophy). Friedel Pick (Deutsche Zeitschrift für Nervenheilkunde, Vol. 17, Nos. 1 and 2, p. 1).

A man, 52 years old, said that he had been healthy until two years previously, when he suffered from articular pain in the lower limbs, and had weakness of the upper and lower extremities and of the neck musculature. When he was examined the atrophy was quite marked, especially in the sterno-cleido-mastoid muscles. The thenar and hypothenar eminences were also much wasted. Sensation was not disturbed, reflexes were feeble, and there were no fibrillary tremors and no reaction of degeneration. A year after this examination the speech was found to be much disturbed, and fibrillary tremors of the tongue were observed. The case was believed to be one of spinal muscular atrophy. A necropsy was obtained and the spinal cord, brain and peripheral nerves were found to be intact, except the spinal accessory nerve. "Simple" atrophy with lipomatosis was found in the muscles. The post-mortem findings showed that the case was one of muscular dystrophy.

Weakness and wasting following articular pain in the lower extremities seemed to indicate that the case was one of polyneuritis, but against this diagnosis was the absence of sensory disturbances and of reaction of degeneration, although quantitative changes were observed. The case could hardly be considered as one of neurotic muscular atrophy. The short duration and the mode of commencement of the atrophy, the age of the patient, the striking atrophy of the small muscles of the hand, the escape of the trapezius, and the disturbances of speech, seemed to make the diagnosis of muscular dystrophy improbable, and that of spinal muscular atrophy more probable.

Pick discusses the significance of the muscle spindles and the Renault's bodies in nerves. The degeneration of the spinal accessory nerve was believed to be secondary to that of the atrophy of the sterno-cleido-mastoid muscle.

The age of the patient—50 years—at the time the muscular dystrophy began was noteworthy. The appearance of this disease after the age of forty seems to have been observed only by Landouzy and Dejerine, Erb, and Linsmayer. The involvement of the small muscles of the hand in the early stages of dystrophy, as in this case, has been observed only a few times. The intense atrophy of the sterno-cleido-mastoid muscle, with relative integrity of the trapezius, was also unusual. Pick refers to much of the literature on the subject of muscular dystrophy, and comes to the conclusion that muscular dystrophy should be regarded as a primary myopathy. SPILLER.